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THE UNIVERSITY OF TORONTO MEDICAL JOURNAL

(An Undergraduate Publication)

This journal is published seven times a year, November to May, by the Medical Society of the University of Toronto, to serve as a medium for student expression and to bring to the student recent articles of scientific interest.

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EDITORIAL

"THOU HAST APPEALED UNTO CAESAR . . ."

". . . unto Caesar shalt thou go!" But there are ways and ways. Some letters written to The Varsity by certain Medical students in the early part of this year bring up a point which is evidently not well understood by the student body as a whole. We refer to criticism on the part of students, of the methods of teaching and other relations between the Staff and Undergraduates. Most Departments welcome student criticism if it is presented in a dignified manner, especially if it is of a constructive nature. In all affairs concerning the Medical student body as a whole, the Medical Society represents this body. Therefore, any criticism should be referred directly to the Medical Society Executive, who will act upon it as they see fit, addressing themselves to the Faculty through the medium of the Assistant Dean and Secretary, Dr. Ryerson. A question which concerns one Year only should, of course, be brought to the notice of the Year's Executive, who may bring it before the Medical Society Executive. We can almost hear someone murmuring "Red Tape," but it must be agreed that such a course as outlined would be the most diplomatic and the best way to avoid such undignified action as has lately been evidenced. Under

our present system of government it has always been the privilege of any citizen to appeal to authority if he does not like any part of the established order of things. In Medicine, we consider the opinion of even one Sixth Year student to be worthy of consideration, but the publication of his opinion to the student world at large with the supposed intent of rallying supporters to his cause we consider unnecessary and in very poor taste and probably also as detracting from the value of that opinion.

THE BURNSIDE FEE.

Several years ago, before the organization of the Faculty of Medicine on its present basis, students in Medicine were required to pay certain fees to each of the Hospitals at which they received instruction. At the present time all these fees with the exception of the fees for the Burnside Hospital, have been incorporated into the annual fee which is now paid to the University of Toronto. To quote the Calendar of The Faculty of Medicine, "The composite fee of $200.00 includes one Session's clinical facilities at the Toronto General Hospital, St. Michael's Hospital or the Toronto Western Hospital, and the Hospital for Sick Children, but does not cover the midwifery ticket for the Burnside Lying-In Hospital, which must be paid in addition, to the Bursar." This $8.00 fee must not be regarded, therefore, as a fee for three day's stay in this Hospital without meals, but rather as a fee for all the clinical instruction received in a five-weeks' course at this Hospital. Students may therefore find it more desirable to consider the fee for tuition in the Sixth Year as $208.00 rather than $200.00, as in other years.

THE AIM OF THE JOURNAL.

The aim of the Journal, as may be seen from the title page, is two-fold: To serve as a medium for student expression and to bring to the student recent articles of scientific interest. This calls for a two-fold response on the part of the student in support: Subscription to the Journal and writing articles to submit to the Journal for publication. A few years ago serious consideration was given to the question whether or not the Medical Society fees should be raised by $1.00 and every student be entitled to receive the Journal automatically. This was not carried out, chiefly because not a high enough percentage of students already subscribed to the Journal. At the same time it was pointed out what value the Journal could be to the students, particularly to those of the upper years, but also in time to those of the lower years. It has always been the policy of the Journal to publish articles which reflected present established conceptions of clinical topics as taught in Toronto, and present methods of treatment as practised here, rather than devoting very much space to new, untried ideas and methods of treatment of disease. In this way, the subscriber is presented with numerous articles in which various clinical topics are treated in their entirety, the material being drawn from several sources, condensed and arranged in such a form as to give a comprehensive description which, it is considered, will aid the student tremendously in his studies. The value of such presentations in forming a ground-work on which to base a more detailed study of a subject, or for review of a subject at examination time, is obvious. Many a time have we heard students of the upper years remark how useful the Journal has been to them in their studies; more often have we heard the wish that they had subscribed to the Journal right from the First Year, and had saved their copies. We cannot too strongly urge the students of the lower years to begin subscribing to the Journal right from the start. This must not be considered as advertising or as a means of boosting subscriptions, but rather as advice which we feel it our

duty to give; for the Journal is published by the Student body as a whole through the Medical Society and we, the Editorial Staff, are the spokesmen for this body.

By the same token, it is not up to the Editorial Staff alone to see that the Journal maintains the high standard set in previous years, it is again up to the student body. Most of the student articles received by the Journal are primarily written for some other purpose, either as theses, or as papers to be delivered at meetings of AOA, the Hippocratic Society, the Physiology Journal Club, etc. May we repeat the old aphorism that the one who prepares the paper derives more benefit from it than he who hears it delivered or who reads it. We use this as a plea for more student articles written primarily for publication in this Journal. Many hospitals enquire from applicants for internships and other positions as to what articles they have written. Here is your opportunity to start. In addition to the customary descriptions of definite clinical conditions, reports of interesting cases seen on the wards and reports of interesting cases seen at autopsy are always acceptable. Also articles concerning the history of Medicine and articles on subjects met with in the basic sciences of Medicine,—Chemistry, Biology and Physiology, etc., are greatly to be desired, and members of all years should be able to write something of interest for publication. Incidentally, let us have a little humour in the Journal this year. Do not submit any plan or fancy jokes, but let us hear any of the amusing incidents and interesting notes with reference to the history of Medicine which may be found.

"A little nonsense now and then
Is relished by the wisest men."

CRITICISM.

We are always striving to improve this Journal; may we have your strictest criticism! Make it constructive if you can but even the hardest knocks will be cheerfully received and we will try to turn them to good advantage. Letters received by the Editors may be reprinted either in part or in toto in the Journal, but we most emphatically do not intend to open a correspondence column.

STAFF ARTICLES.

We are always delighted to receive any articles written by members of the Staff, for publication in this Journal. If any member of the Staff has an article already written which would be suitable for this Journal, or an article in preparation or in contemplation, we should be glad if he would submit it to us, provided that its publication in these pages would not jeopardize its reception elsewhere.

Medical-at-Home

Supper Dance

ROYAL YORK HOTEL

January 26th, 1934

Wright Bros. and Their Orchestra ∴ Dancing 9-3

DR. WILLIAM HOWARD DICKSON

DR. WILLIAM HOWARD DICKSON.

RADIOLOGY in Canada lost one of its brightest minds, the medical profession a valued leader, and students of medicine a beloved friend and teacher with the passing on the 30th October last, of William Howard Dickson, M.D.C.M., F.R.C.P. (Canada), member of the Royal Society of Canada. At the early age of 55, he was the victim of cardiac trouble, the original onset of which occurred in February, 1932.

A native of Pembroke, Ontario, Dr. Dickson was the only son of the late Dr. William Welland Dickson, of the same town. He graduated from McGill University in 1904, and almost immediately left for the Pacific Coast, where he engaged in general practice at Anyox and Phoenix, both mining towns.

An early appreciation of the growing importance of the X-ray in diagnosis led Dr. Dickson to devote his life to the new science of radiology. A staunch disciple of Dr. Lewis Gregory Cole, of New York City, he developed the use of the rays in the diagnosis of gastro-intestinal pathology to an extraordinary degree of accuracy. Students who attended his lectures, as well as physicians who referred their cases to him, will readily recall the mastery of this diagnostic field he had achieved.

"In his work," says Professor Goldie, "he was our leading light; with his simplicity and intellectual honesty, that clear-acting brain brushed aside the unessentials to point out the way or face the fact with 'We do not know.'"

It was his frankness and honesty, indeed, together with an infectious enthusiasm for his chosen work, that made him so inspiring a teacher. Not without significance is the fact that the six active pallbearers who bore him to his rest were all junior men, with whom he had so genially and patiently carried on research work of more than ordinary importance. He had, to quote Professor Goldie again, the happy faculty of "smoothing the way of his elders, and inspiring his juniors with a sympathy and tact that brought out their best."

His recent pioneer work on the use of thorium dioxide as a means of enlarging the diagnostic field of radiology is well known. But it was Dr. Dickson's diagnostic ability — almost oracular — in the gastro-intestinal field that brought his most substantial and widespread renown.

He was a tireless worker, a keen student, an original thinker. And of his many activities, none was regarded more earnestly, or more worthy of thorough preparation, than his teaching. Yet Dr. Dickson never referred to his lectures to students—it was always his "talk to the boys."

Percy Ghent.

RHEUMATIC HEART DISEASE*

F. J. HOSKING, '34.

History.

Edward Jenner in 1789 was the first person to draw attention to the fact that there is a relation between Rheumatic Fever and Heart Disease. Bouillaud, in 1836, published papers showing that inflammation of the heart associated with acute rheumatic fever is the rule rather than the exception. He also goes further, saying there is permanent valvular damage following an attack of this disease.

Incidence and Etiology.

These topics have been dealt with in the previous paper.

Morbid Anatomy and Histology.

Gross Changes.—(a) At post mortem the heart will have a greater width due to dilatation of the ventricles, accompanied by a thickening of the ventricular wall. Secondary to this is a dilatation of the auriculo-ventricular rings, which are slung in the muscle of the heart. The arterio-ventricular rings do not dilate, due to their fibrous nature.

(b) When residual cicatrical phenomena of rheumatic carditis enter the picture, there is a great hypertrophy of muscle.

(c) The myocardium in the large majority of cases shows no gross lesion. Other findings are inconstant, such as—1. White flecks of fatty degeneration. 2. Spots of fibrosis which are scars. 3. Increase in fibrous tissue about valve rings and at the apex of papillary muscles.

Microscopic Changes.—(a) There are found characteristic submiliary nodules (Aschoff nodules), made up of a fibrous matrix, certain large cells, fibroblasts, plasma cells and lymphocytes. These nodules are most common in the interventricular septum and in the muscle of the left ventricle.

(b) Changes in capillaries and arterioles are most marked. The endothelium proliferates causing stenosis, not seen in the larger vessels, and circles of fibrosis often form sleeves about vessels.

(c) Muscle cells show a fatty change often associated with shrinkage of the cell.

Endocardium.—The mitral valve is injured in 100% of cases, the aortic valve in 50%, and the tricuspid in 35% of cases. In the acute condition there are fine fibrinous deposits, pinhead in size, on one side of the valve curtain, which is thickened and pinkish in colour instead of showing its normal pearly sheen. Microscopically the valves show proliferation of fibrous tissue, ingrowth of vessels with a stenosis of the same, submiliary nodules, and surface necrosis with neutrophiles and fibrin plastered upon it.

Mitral Valves show—1. Very thick curtains. 2. Stiffening. 3. Drawing of curtains up toward auricle. 4. Deposition of calcium salts. These conditions may produce an aperture which is a mere slit between stiffened curtains, resembling a button-hole, dilatation of the valve being due to dilatation of the heart.

Tricuspid Valves show similar lesions not so marked.

The Aortic Valve is more often calcified, its three cusps being always involved together.

Pericardium.—There is pericardial involvement in all patients under 10 years of age, in 85% of patients under 20, and in 53% of all patients. The

*Presented before the Hippocratic Society, February, 1933, in conjunction with the preceding paper on Rheumatic Fever by L. N. Pearlman.

first changes are a slight reddening and roughening of the serous surface, with an increased turbidity of the pericardial fluid. Severe cases may show large masses of pinkish fibrin filling the pericardial space. Pericardial effusion occurs in 10% of cases, but the amount of fluid is rarely large.

Aorta.—Occasionally, small pale yellow subendothelial patches may be seen on the intimal surface.

Morbid Physiology.

Mitral Valve.—This valve has two functions, to prevent regurgitation and to allow free passage of the blood into the ventricle. Mitral regurgitation is much more common in young patients than in old, showing that dilatation of the ring causes more regurgitation than shrinkage of the sclerosed curtains. It is, however, reasonable to believe that the nature of the murmur, i.e., its loudness or softness, is due to the hardening of the curtain. When the valve is diseased by the chronic condition it becomes hardened, and obstructs the flow of blood, producing the presystolic murmur.

Sounds — The accentuated first sound in the mitral area owes its origin to hypertrophy of muscle. Later in the disease the first sound becomes banging in character, due to the left ventricle contracting on a small quantity of blood; the stenosed valve preventing proper filling. The first sound is partly due to the valve cusps coming under tension. Due to the hardened sclerotic nature of the diseased valve, it comes under tension much more suddenly, thus the snapping nature of the first sound. Reduplication of the second sound at the apex is a fairly common feature in rheumatic patients.

Adventitious Sounds — There are three types of mitral murmurs: 1. The characteristic murmur, which is short in duration and runs up to the first sound, and is known as the presystolic murmur. This is due to the contraction of the auricle forcing blood through a stenosed opening. 2. A murmur may start in mid-diastole and run, not changing in nature, until ended by the first sound. This is due to loss of function of the auricle. 3. A murmur which starts just after the second sound, lasting a very short time, is due to blood passing through a stenosed opening into the relaxing ventricle.

Tricuspid Valve.—Functional lesions of this valve are too rare to diagnose. Stenosis of this valve has, however, been known to cause obstruction.

Aortic Valve.—This valve also has two functions. The prevention of regurgitation is the one most often lost in rheumatic heart disease. Regurgitation is due to the damaged cusps, and not, as in syphilis and atheromatous degeneration, to dilation of the valve ring. Stenosis of the aortic valve is relatively uncommon.

Pericardium.—Pericarditis causes embarrassment when a semi-solid exudate becomes organized, binding the pericardium to the heart. The embarrassment is still more pronounced when adhesions form to pleura, mediastinal fibrous tissue and chest wall, etc. When the heart enlarges in an acute attack it often causes great distress.

Myocardium.—In acute attacks there is a loss of tone and power to contract. Thus rapid ventricular dilatation follows, with a fall in blood pressure, muffling of cardiac sounds and presence of triple rhythm. A late disturbance is auricular fibrillation. This is due to the damaged auricular muscle forcing the impulse to seek undamaged muscle paths on which to travel, until it finds its way into a circus movement.

Symptoms.

Dyspnoea.—Is the most common symptom. It can be due to mitral stenosis, mitral regurgitation, pulmonary valve insufficiency, muscle degeneration or infarct of lung caused by emboli.

Cyanosis.—Goes hand in hand with dyspnoea. There is a heightened col-

our over the malar prominences. It must be remembered that cyanosis can be also due to a poor ventricular muscle failing to send the blood through the peripheral circulation.

Cough.—Where mitral obstruction is present, the bronchi becomes very susceptible to bacteral irritation. A morning cough is often described in these cases.

Haemoptysis.—From this cause comes second in frequency to that produced by tuberculous infection.

Pain.—There are two types common in acute attacks. 1. That due to pericardial inflammation, referred to the sternum and precordium, epigastrium, and occasionally to the interscapular region. 2. The angina type of pain which may be brought on by exercise, a chill, lying down, etc. A dull ache may be a symptom in acute or chronic dilated hearts. This might be explained by stretching of the pericardium.

Physical Signs.

There may be projection of costal cartilages from the second to the fifth in young patients. Respiratory movement may be inhibited by pericarditis and there may be abdominal rigidity. The visible cardiac displacement when downwards means hypertrophy, when outwards means dilatation. The visible pulsation may disappear in an acute attack. Increase in area of pulsation means dilatation, hypertrophy, or both. Pulsation in the third left interspace is produced by enlargement of the whole heart and by enlargement of the left ventricle. Pulsation of the aorta in the second and third right interspaces is present in regurgitation with a large output of the left ventricle.

Palpation.—Apex beat. In mitral stenosis the beat comes with a bang sharply and suddenly against the chest wall, and it also sinks away suddenly. *Thrills.*—1. Systolic. At the apex a thrill is due to mitral incompetence; at the base, to aortic obstruction. Aortic thrills tend to radiate to the right clavicle. 2. Diastolic. Two thrills are felt, just medial and above the area of maximum impulse. The early diastolic is fine, diminuendo in character, and persists. The late diastolic or presystolic thrill is gross, crescendo in character, and ends at the first sound.

Pericardial friction is sometimes palpable.

Percussion.—Enlargement of the heart may be found. Dullness at the base of the left lung is seen in acute attacks and is due to reflex collapse.

Auscultation. — Heart Sounds. A sharp snap is the characteristic nature of the first sound in mitral stenosis. The first sound is loud, deep toned, and muffled where there is much hypertrophy of muscle. This is most often marked in aortic regurgitation. A combination of these types of sound is not infrequent. Accentuation of the pulmonary second sound is common. The aortic second sound is accentuated where hypertension is present, and where this occurs a possibility of nephritis must be thought of. Rarely is the aortic sound accentuated in rheumatic carditis. A softening of the first sound at the apex is due to pericardial effusion or to very weak muscle. The former is rare and the latter is present in acute conditions.

Adventitious sounds.—Apical systolic murmur varies in intensity with the heart sound and in about sixty per cent. of the cases disappears as a presystolic develops. This is explained by dilation of the ring in the earlier cases with regurgitation. Later when the cusps are hard and drawn together regurgitation is not allowed, but stenosis is present. Aortic murmurs, systolic in time, do not mean obstruction unless a regurgitant murmur or other signs and symptoms of aortic incompetence are present. *Diastolic murmurs.* The presystolic murmur is the one which most often indicates anatomical lesion. Its characteristics are—its position, the way it runs into the first sound, it is heard over a limited area (mitral area), coarse in character, crescendo in nature. A

presystolic murmur is best heard with the patient lying on the left side and is often associated with a systolic murmur. An early diastolic murmur may be heard at the apex, more frequently in earlier cases of rheumatic heart disease. It is produced by a relative stenosis of the mitral valve. Regurgitant murmurs at the base are more frequent in males than in females, and chiefly after thirty years of age. They are most often heard along the left margin of the sternum and are soft in nature, but may be harsh.

Pericardial friction—Is heard in the more or less acute stages of the disease, usually in patients under thirty years of age. Only 10 to 15% of patients ever shown this sign. The area at which the rub is heard does not extend beyond the precordium, the sound is soft and brushing in nature to and fro in character with a superficial quality in 9 out of 10 cases.

Pulse Arrhythmia. — A quickened pulse is present in the active stage of the disease, especially in myocardial failure. Paroxysmal tachycardia lasting a few minutes to a few days may be found. The attacks are brought on by emotion, work, digestion, etc. The rate is usually a definite multiple of the rate in the intervals. An abnormally slow pulse in this disease is a rarity. Its cause is usually due to extra-systoles. Premature beats occur in 5 to 10% of cases mostly in the later stages of the disease and are thought to be due to plugging of the smaller arteries. Total arrhythmia is the most important heart irregularity in rheumatic carditis. It occurs in 10% of cases. The muscle injured by the disease produces the auricular fibrillation. When this occurs the patient steps down to a lower plane of activity. The heart rate is usually about 140 with a pulse deficit. The worse the condition of the muscle the greater the pulse deficit.

Blood Pressure.—The blood pressure is usually low in acute cases. Where the blood pressure is high it is most likely that nephritis is an influencing factor. Great pulse pressure is associated with aortic regurgitation, an enlarged ventricle and capillary pulsation.

Diagnosis.

In making a diagnosis there are three things to consider. 1. Is the case one of organic heart disease? 2. Is the disease rheumatic? 3. If rheumatic, what is the extent? Twenty-five per cent. of cases do not have any symptoms of rheumatic fever, such as arthritis, chorea, etc. When there is a great increase in cardiac dullness, pneumonia may be diagnosed. Pain of acute carditis may be referred to the epigastrium. Patients often do not give an accurate history of early attacks of rheumatic fever, such as growing pains, etc. A diagnosis of the disease is made where there is a true presystolic murmur, where the point of maximum impulse is out, where the border of cardiac dullness is out, when accentuation of the first sound at the apex is present and where there is increase in force of impulse.

Evidence of active infection.—The younger the patient the more active is the disease. Below 15 years the disease is always more or less active, over 30 years the reverse is believed. In children, symptoms are more important than signs. A sustained rise in temperature which is not too high is the strongest point in favour of active infection. Progressive loss of flesh and loss of colour are also indications. Where an ulcerative condition is imposed on a rheumatic, signs of embolism occur. Clubbing of the fingers along with enlargement of spleen is always diagnostic. All attacks of severe carditis have their pericarditis. Signs of active carditis are—tenderness over precordium, catchy pleuritic breathing, dysphagia, immobility, resistance and tenderness of epigastrium. A few chest complications that may arise from rheumatic carditis are: 1. Pleurisy; 2. Pneumonia; 3. Basal lung collapse; 4. Acute oedema. Size and shape of liver may indicate active carditis. It

may be tender and pulsating with rounded border. Ascites is often a sign of carditis. Kidneys are often water logged, with low output of highly coloured urine, albumen being present.

Course and Prognosis.

Of a large number of cases followed by Carey Coombs he found the average age of onset of carditis to be just above ten years. Five per cent. died during the first year, 11% within five years, 21% die within ten years. Where definite signs of rheumatic heart disease are present the average expectations of life are as follows:

Recovery in 31%.
Death before the age of 20 in 28%.
Death before the age of 30 in 38%.
Death before the age of 40 in 50%.

Nineteen per cent. of the patients who survive are cardiac cripples.

Occupation.—Does not seem to influence the condition, unless the occupation produces severe shortness of breath.

Pregnancy.—The effect of pregnancy on a diseased heart does not seem to be very marked. Once in a while it demands too much of the damaged heart and permanent disability remains. Again the patient may not be able to live to the end of term.

An infection of cardiac tissue by a rheumatic disease may be compared to a fire that may die down but never goes out, rising from time to time with sullen heat but little or no flame.

Prognosis of the individual depends on the presence of active infection with the degree of body saturation. On one hand, a child may be killed outright, the illness lasting only a few weeks. On the other hand, there may be no symptoms nor signs for years. Oedema in a child deserves a prognosis of only a few weeks, in a chronic adult, a few months.

Pulsating liver, small renal output, albumen without casts and brisk haemoptysis are very bad signs. Total arrhythmia is of grave significance with an average prognosis of two years, the extremes being roughly one to ten years. The earlier it sets in ,the shorter its course. Extra-systoles when mitral obstruction is present indicate impending fibrillation. Pulsus alternans is the forerunner of catastrophe. Pericardial friction is a bad sign because it indicates massive involvement of heart muscle.

Treatment.

The bodily exertion of the patient should be restricted according to the severity of the disease. The child who is thought to have any carditis should be kept in bed a week. A patient who has active signs and symptoms should remain in bed until all such signs and symptoms have disappeared. In the acute stage the patient should be fed a high caloric diet. A policy of supervision should follow an attack, signs of a recurring attack being watched for. Excessive exercise, fatigue, exposure to wet and cold must be avoided.

Drug Treatment.—Large doses of sodium salicylate are given in acute attacks; a common procedure is to give 20 grains every hour for 8 to 10 hours, toxic symptoms being watched for. Equal quantities of sodium bicarbonate are also given. About a hundred grains of each, given in 4 doses during the day, seems to be effective. This should be continued until active infection has been absent for at least a week, and then the dose should be reduced to 15 to 20 grains a day for a long time.

Morphine or codeine may be given to control pain.

For auricular fibrillation, digitalis is always indicated. Fifteen minims of the tincture every 4 hours at the onset is the minimum. Two or three drams may be given in the first 24 hours with good effect. Quinidine sulphate has a good effect in 30% of patients. Five grains are given every 4 hours. If not a success the treatment should be changed within a week. It is perhaps best to slow the heart first with digitalis.

In progressive ventricular failure,

diuretics should be given and paracentesis done if necessary.

All patients should be fed well and their bowels kept working freely.

Tonsillectomy is advised by some as a routine measure, but this should not be done while any fever is present.

INDICATIONS FOR RADIATION.

The following is an extract from an article in the Radiological Review, October, 1933, on "Radiation Therapy in Medicine," by Ira I. Kaplan, B.Sc., M.D., Director, Division of Hospitals, New York City.

Radiation is useful in the following conditions:

As an emergency measure for enlarged thymus, ideopathic haemorrhages, and where new growths press on some vital structure, such as the trachea, restricting respiration.

In benign lesions such as

A. Dermatology.
   1. Acne.
   2. Eczema.
   3. Sycosis.
   4. Lupus.
   5. Haemangiomas.
   6. Keloids.
   7. Pruritus.
   8. Kraurosis Vulvae.

B. Neurology.
   1. Neuralgia.
   2. Sciatica.
   3. Syringomyelia.
   4. Pituitary Tumours.

C. Gynaecology.
   a. Functional.
      1. Amenorrhea.
      2. Dysmenorrhea.
      3. Metrorrhagia.
      4. Menorrhea.
   b. New Growths.
      1. Fibroids.

D. Medicine.
   1. Tuberculous Adenitis.
   2. Hodgkins' Disease.
   3. Leukemia.
   4. Agranulocytosis.
   5. Hyperthyroidism.
   6. Unresolved Pneumonia.

E. Surgery.
   1. Unhealed Bone Sinuses and Soft Tissue Sinuses.
   2. Adenitis.
   3. Post Operative.
   4. Prostatic Hypertrophy.
   5. Vesicle Haemorrhages.

Conditions so treated are: Superficial malignant lesions of the skin and mucous membranes wherever situated throughout the body. Malignant lesions of brain, head, the oral, nasal and pharyngeal cavities, the oesophagus and gastro-intestinal tract, the respiratory system, the genito-urinary system, the reproductive and genital organs, the breast, the skeletal system, and in cases of generalized malignant metastases.

In some of these lesions radiation is used as the treatment of choice, in others as an excessory agent along with surgery. For epitheliomata of skin, radium acts rapidly and effectively. In cancer of cervix, X-ray and radium together have replaced surgical procedures, while in antrum malignancy, surgery plus radium and X-ray therapy is the accepted method of treatment, and for generalized metastases and advanced lesions, X-rays act as a beneficial palliative.

Sarcoma of all types are best treated with irradiation.

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In looking over a number of hospital records of acute nephritis in adults for the purposes of this paper, it was interesting to find that the great majority had as their onset the "common cold," described in many histories as nasopharyngitis. In the records of children with nephritis there are, no doubt, many more instances of preliminary illnesses of a more extensive nature, such as bronchopneumonia, influenza, scarlet fever, and diphtheria, but in these latter the upper respiratory passages are always involved. It is to these upper respiratory passages with the streptococcus as the primary or secondary invader, that every possible attention should be paid, in the prevention and treatment of nephritis. It is the recurrence of the acute process in the glomeruli that either gradually lowers the permanent nephritic or, by its severity, suddenly plunges the advanced chronic case from tolerable health into fatal uraemia. To prevent respiratory infection, measures advised, such as avoidance of crowds during the prevalence of "colds," avoidance of exposure to chill, may be scoffed at in health. They should be taken seriously by the individual who has albumin and casts constantly present in the urine. It goes without saying that acute infections of the nose, throat and mouth should have the most thorough palliative care. A little later areas of residual infection must be sought out and this search should always include the nasal sinuses and the adenoid tissue of the nasopharynx, as well as the tonsils and teeth. When such foci of infection are discovered during the course of an acute nephritis, an important decision that has to be made is, at what juncture to advise interference. Prolonged and severe reactions in the glomeruli, with increased haematuria, may follow operation. For this reason, in the Toronto General Hospital the policy is to delay tonsillectomy, radical operations on the antrum, etc., until the patient is not only well, but has become completely free of albumin and red cells in the urine, a course which may call for a waiting period of two or even three months. This plan is favoured by Mosenthal, who writes the Oxford Monograph on nephritis, and who in his wide survey of the experience of different clinics has to report the frequent occurrence of long, severe reactions, but reports no fatal ones. That reactions are not inevitable is proved by the success which has attended earlier operation in the Hospital for Sick Children, where the policy is to act as soon as the general condition of the patient warrants such. Sometimes circumstances make the decision. Thus one case stands out in the writer's memory. A young adult had persistent red coloured urine for days. An erythema, with intolerable itching, added such misery to the situation, already bad enough with a rigid milk diet for oedema, that interference seemed imperative. A long operation under ether anaesthesia, during which many isolated tonsil remains were dissected out, was not only well borne, but the colour of the urine changed at once and recovery was rapid. Decision on this important point is difficult, but the weight of opinion rather favours conservatism.

Acute Nephritis and Acute Exacerbations in Cases With Permanent Renal Changes.

The first opportunity for action is often the convulsive state. This is now felt to be due to oedema of the brain,

therefore the principle of treatment should be to remove water from the system. Magnesium sulphate, acting as it does by osmosis, is the most commonly used remedy. As neither the salt itself nor the accompanying water are absorbed from the intestinal tract, it may be used very freely. Every case of early nephritis should at once be given from one half to one ounce of magnesium sulphate in an equal quantity of water daily. When a severe headache or a rising blood pressure foretells an approaching convulsion, or when a convulsion has occurred, administer by mouth, if possible, every four to six hours should be started, together with twice the amount by rectum at the same intervals. Since the introduction of the plan by Blackfan, the intravenous use of a one per cent. magnesium sulphate solution, given at the rate of 2 c.c. per minute until the blood pressure has fallen to 120 m.m., or until 15 c.c. of the solution for each 2.2 pounds of body weight has been given, is the routine procedure in the Hospital for Sick Children. This is added to the oral and rectal plan. An alternate plan to this last is the intravenous use of glucose 50% solution in amounts up to 3 ounces twice or three times in twenty-four hours. Another plan for relieving the oedema of the brain is spinal puncture. Elwyn, in his monograph on nephritis, very strongly recommends this, either whenever a convulsion is seen to be "working up," or has just quieted down and may recur. This is also done as a routine plan in the Hospital for Sick Children, especially at is includes the diagnostic help of ruling out convulsions from head injuries. For private practice where facilities are limited, the immediate management of a convulsion is to start the inhalation of chloroform and continue it until a hypodermic of morphia has been given and has taken effect. Then magnesium sulphate should be given, or if the patient cannot swallow, a drop or two of croton oil in paraffin oil or butter should be placed on the tongue. Arrangements for rectal or intravenous treatments should then follow.

In the dietetic management of acute nephritis, there are two plans to discuss. The one plan, roughly speaking, is to treat the patient, the other is to treat the kidney. The former is the one favoured by prominent books of the day. As it is now known that the oedema of acute nephritis is not due to the state of the kidney itself, and that the convulsive phenomena of the acute process are probably due to oedema of the brain, and certainly are not related to uraemia or to nitrogen retention, and as moreover, the alarming symptoms of acute nephritis die down in the majority of cases in a few days, it is not considered essential to treat the kidney very definitely, but rather to build up the patient. Thus, Mosenthal, in the Oxford Monograph, rather as a concession to former ideas, advocates only a week of a low protein diet allowing fats, sugars, fruits and low grade vegetables cooked without salt, in any quantity, and is willing to add a quart of fluids in the form of weak tea or coffee, fruit juices, water, even in the presence of some oedema, restricting fluid only in the presence of irksome oedema. At the end of one week, a gradual return to normal diet is advised, provided the haematuria, oedema, and hypertension are on the wane.

In the Toronto General Hospital for the last ten or more years, a very different plan has been applied by Dr. W. R. Campbell, who has charge of cases of nephritis. His plan is to treat the kidney first and foremost, and to let nature take care of the patient. He feels that while, as is commonly pointed out, the acute process dies down quite rapidly, yet a needlessly large percentage of cases, while recovering their well-being, still show albumin and red cells in their urine. Unless these have disappeared completely, the lesion is not healed, and a miserable future of chronic nephritis awaits the patient, in all probability. Therefore,

a rigid plan is carried out until the urine is quite normal or until so many weeks have gone by without improvement that this hope has to be abandoned. By choosing milk as the staple article of the plan, it is possible to accomplish some definite purposes. It is known precisely what the patient is getting at all times. It is possible to easily regulate the exact amount of protein, salt and fluid ingested. There is not a variety of mineral salts to be excreted, such as exist in fruits and vegetables. The kidney is spared work by keeping the body metabolism low by a meagre caloric intake while the patient's strength is spared by continued bed rest. Orange juice is added for its vitamin content. Twenty to twenty-five ounces of milk with one orange is the total daily allowance for cases of moderate severity. A number of histories show that this amount has been the sole intake for three weeks at a stretch, supplemented sometimes by chipped ice to suck. With diminution in the amount of red blood cells in the urine, this amount is increased to thirty or at most, forty ounces. On this latter amount, many of the cases have been kept for one, two, and in one instance at least, three months. If a flare-up of haematuria occurs, the amount is reduced to twenty ounces for a period. Encouragement as to their progress leads patients to put up with this austere plan. Arrowroot biscuits, a baked potato, a little cream or lactose in fruit juice are occasional additions to the milk. Not until red cells have practically disappeared from the urine is a high carbohydrate diet allowed and not until albumin has cleared up is there a return to full diet. This plan is in marked contrast to the alternate one more generally employed in that the amount of protein is never more than 40 grams (contained in one quart of milk) so that the patient is held just within a nitrogen balance, whereas in the more liberal plan previously described it is urged for the sake of the patient as a whole to advance the protein to 60 or 70 grams or more at the end of a fortnight. Milk is chosen not with any idea that its form of protein is eliminated by the kidney with auy less work than the protein of meats, but because the amount of salt and fluid accompanying the protein of milk is so easy to compute. Moreover, the thirst caused by meats or fish, calling for more fluid allowance, does not apply in the case of milk. Dr. Campbell feels sure and has convinced those about him that in acute nephritis by this means he has achieved perfect results in cases which would have passed into permanent damage on a more complacent management. In the Hospital for Sick Children it is found that a ketogenic acidosis accompanies a low intake, and therefore fluids given freely and high carbohydrate intake become necessary from the first.

Drugs, with the exception of magnesium sulphate and digitalis for support to the heart which is sometimes affected in acute nephritis, are contra-indicated. Diuretics will more likely cause a diminution than an increase in flow. Even in persisting oedema it is wiser to wait, keeping the patient on twenty to thirty ounces of milk alone.

Physical agents such as sweating, hot poultices to the loins, hot rectal irrigations have been abandoned as being useless in purpose and debilitating and irritating to the patient.

Decapsulation of the kidneys would seem to have no role for the anuria or oliguria of acute nephritis in the light of modern knowledge. Mosenthal says it should never be done in nephritis. It may have a doubtful place in chronic oedema cases or in passive congestion of the kidney. The writer took part as an intern in 1902 at the old General Hospital in the case of a man who underwent double decapsulation for oedematous nephritis of some months' standing. In 1930 this individual was alive. He dates the disappearance of his troubles from the operation, but has spent a life time of semi-invalidism. It is hard to say what part the surgical

procedure played here, as operation was followed by a long stay in bed in the hospital.

.. **Subacute nephritis**: This form of the disease, hastening the patient to death in a few months from the blended effects of a severe persisting acute process and a rapidly induced chronic change, has to be managed as well as possible by methods suitable to both phases.

Subchronic or second stage nephritis: Patients in this group show albumin and casts in the urine permanently. If infection is latent, they may be free of clinical symptoms. If it is smouldering or recurring at intervals there will be disturbance in salt and water balance and long-continued oedema may be the outstanding feature. Nitrogen retention is not a problem as yet, and may not be for several or for many years, even twenty or more in some instances. These so-called "latent" cases are often discovered during a routine examination for life-insurance or other purposes. In them, infections should be constantly sought. But, alike with patients suffering from high blood pressure, they should not be subjected to the rigid protein restriction practised during the last few generations, or the rigid salt restriction of the last generation. This dreary plan wherein all flesh food, and eggs, and latterly salt, have been forbidden, defeats its own ends by producing anaemia, debility, and lowness of spirits. In recent years much interest has been taken in the effects of high meat diets on the kidney. The Eskimo, who consumes probably three times as much flesh as the average individual of other races, shows no tendency either to nephritis or high blood pressure. Stefansson lived for nine years on a meat and fat diet without harm. In Newburgh's laboratory, a worker ate daily for six months thirteen ounces of beef liver, ten ounces of veal, twenty-three ounces of beef tenderloin, and three ounces of dry beef without any damage. This was a total of eleven ounces (340 grams) of protein, whereas three or four ounces is probably a fair average consumption in general. There seems, therefore, to be no justification for the fear of the proteins of meat. There seems also no reason for restricting protein food to a low level during this phase of nephritis wherein functional tests show excellent nitrogen elimination.

In subchronic cases with lengthy and intractable oedema, there is reason for maintaining protein at an average normal level, and in the severe oedematous picture called nephrosis, Epstein's plan of feeding twice the usually consumed amount in the form of meats has improved the well-being of many patients and has apparently done no harm. In all oedema of long duration it is necessary to fear the lowering of the albumin of the blood as one contributory cause, and to meet it by proper protein intake. Dietary restrictions for oedema lie in the salt and fluid department. It is therefore necessary to have some principles for prescribing a regime relatively low in salt. Rigid prolonged restriction is not safe. For moderate cases, omissions of table-salt during meals and avoidance of salted foods and meat soups and gravies will suffice. Next to these, milk, cheese, butter and bread and other cereals, form the principal source of salt in the ordinary dietary. Bread, rolls, and cake may be cooked without salt and salt-free butter used. A further and final step may be to cook vegetables without salt, but if this degree of restriction is applied, there should be intervals of remission. Fluid restriction in oedema must be based on the facts that there is much water in all foods, and especially in the fruits and vegetables, and that, per contra, roughly half of the total water ingested leaves the body via the breath, skin and bowels. In severe oedema the drink allowed may be cut down to twenty ounces, or three cups a day, if the patient is receiving fruit and vege-

tables, so that all channels of elimination may be supplied.

The use of diuretic drugs in persistent oedema presents a strong appeal, but also presents dangers and disappointments. It is probably wiser to use these drugs for short periods only, when successful, and to change to one of the others if an immediate response is not obtained. When the oedema accompanies an exacerbation of the acute process, as evidenced by haematuria, diuretics should not be used, as mentioned previously under acute nephritis. When the oedema appears to be due to cardiac failure, digitalis may be given (thirty minims a day) for several days, followed by one of the so-called dilators of the smaller arteries, diuretin (thirty grains a day), theocin (fifteen grains a day), or euphyllin (ten grains a day). If a result occurs in one day, postpone readministration for several days. If no result occurs at once, change the drug. For oedema where blood cells are not present in the urine, the acid salt ammonium chloride (three drams a day) may succeed. As it operates by producing a mild acidosis, there is some danger that this acidosis may become severe if prolonged. After a few days the ammonium chloride may be accompanied by the intravenous or intramuscular administration of the organic mercurial salyrgan (one or two ampoules twice a week for a short period). Finally, though not of use if cardiac failure plays a part, urea in large doses (one to three ounces per day for adults) may be tried in coffee, fruit juice or water. This is safe provided there is no blood nitrogen retention to start with, and provided by blood examination every four or five days the urea nitrogen of the blood is not raised thereby to exceed 80 mgs. per 100 c.c. Purging with magnesium sulphate is desirable continuously. Sweating measures have been abandoned as useless. Aspiration of pleural fluid is highly desirable. Tapping of the abdomen is doubtful. It appears to become urgent sometimes. Peritonitis often ensues. Some experience has made the writer wary of withdrawing all the fluid from the abdomen at the first tapping. Instead of the expected relief, the patient may sink into fatal coma.

True Chronic Nephritis: This stage first begins when the kidney can no longer handle an occasional excess of nitrogen intake. A period of gradually increasing failure follows, but this period may last over many years. The next phase is when the kidney can no longer eliminate the end-products of the amount of protein necessary to maintain the body in nitrogen equilibrium. Even this phase may last for two or three years, life under these last conditions being maintained by an obligatory polyuria. The treatment of a patient with true chronic nephritis may therefore last over a long time, depending on the presence or absence of infections. As more and more of the

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important kidney substance is lost, debility, anaemia, loss of weight occur. It is therefore necessary to provide a nutritious diet. A moderate amount of nitrogen retention, 50 to 60 milligrams, is better overlooked in favour of adequate nutrition. Compact high-grade protein food such as meats, fish, and eggs are greatly preferable to the monotonous and bulky milk products. The amount of protein in true chronic nephritis, as guided by the feelings of the patient and the functional tests, should range from 70 to 40 grams. The latter is the amount that will preserve nitrogen balance in the average adult when complete renal insufficiency has finally set in. In practice the tables of O'Hare, to be found in the books of the day, provide an easy way of prescribing the desired amount of protein food and a liberal general diet. The first table indicates in household terms units of food; each containing four grams of protein. Thus one unit of meat is contained, in the cut-up state, in one tablespoon. The range of units in Table One includes, besides the various animal products, various articles from baked beans to pie, all of which contain protein of a good grade in some degree. In Table Two is listed the non-protein foods, which may be eaten in any quantity. Salt should be somewhat restricted in chronic nephritis, though oedema is not a feature. If in the latter stages polyuria becomes irksome, this may be alleviated by reducing the protein somewhat, but never by restricting fluids.

Purgation should be constantly maintained throughout the end-stage, to prevent absorption of decomposition end products from the bowel. For terminal uraemia, the only extra contribution possible is the administration of glucose solution freely to combat acidosis and dehydration. This terminal uraemia may be precipitated in a few days at any stage of true chronic nephritis by the recurrence of the acute process from infection. Fluids freely are called for here, even in the presence of oedema.

ARTHRITIC ADAGES.

A joint on the move is worth two in the splints.

An ounce of preventive thought is worth tons of rheumatic cures.

Early to bed and slowly to rise.
Is a rule the arthritic will follow if wise.

Better a dinner of herbs and vitamins which science prescribes than starches and sweets which the joints hate.

Elimination on time saves deposits of lime.

—Copied from the Arthritis Exhibit, Century of Progress, Chicago, Illinois.

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THE PRE-ECLAMPTIC STATE

PART II.

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Pathology.

Because of the absence of autopsies on patients who have died in the pre-eclamptic state, the only available pathology is necessarily that of eclampsia itself. Williams states that the main lesions in eclampsia are found in the liver, kidneys, heart and brain, but that with the exception of the lesions in liver, the anatomical changes are not constant and characteristic.

Kidney.—Although most cases of eclampsia present renal changes when studied in the autopsy room, it does not seem that the renal changes are characteristic of the disease, and are probably more the result than the cause of eclampsia. Furthermore, in patients who recover, the prompt return of the urine to normal indicates that the renal changes must be relatively trifling.

In eclampsia the kidney is not diminished in size; the appearance is anaemic, the cortex cloudy, and in many instances presents small punctate ecchymoses. The capsule as a rule is not adherent.

Microscopic examination shows degenerative changes in the convoluted tubules and the vessels. The epithelium of the damaged urinary tubules is apt to be disintegrated, swollen and deprived of nuclei. The capillary vessels are filled with red cells, showing a marked vascular stasis, and may also contain actual thrombi. The interstitial tissues of the kidney seem to be little changed, and the process involves almost entirely the parenchyma of the organ. Infarcts may be present, and are due to the migration of emboli from other organs.

Liver.—The liver in gross appearance presents irregular haemorrhagic areas, sometimes of large extent. On section there are alternating patches of light and dark areas which give a mottled appearance to the organ. There is necrosis in the lobules and portal spaces, which has been attributed to thrombosis in the small portal vessels with consequent degeneration. This peripheral necrosis of the liver lobule is generally considered a characteristic lesion of eclampsia, though it is sometimes lacking in patients who have died from what was clinically eclampsia.

Brain.—Oedema of the brain, with its resultant intracranial pressure, is declared by Zangemeister to be the cause of eclampsia.

Haemorrhagic lesions in the brain have been noted in most autopsies in eclamptic subjects, both substance and cortex being involved. In addition, diffused areas of cortical softening are frequently observed, as well as oedema or hyperemia, although oedema is probably not reported as often as it occurs because of its rapid subsidence after death. The haemorrhages are probably thrombotic in most instances, but, as Welch has pointed out, the vessels may rupture as the result of the solvent action on the endothelial lining by a circulating toxic enzyme aided by the increased blood pressure. The extent of the bleeding varies from that of small degree in the pons, to an extensive effusion into the lateral ventricles, which distends their cavities and lacerates their walls. True, cerebral apoplexies are also found, usually in the distribution of the middle temporal artery; and in those cases of eclampsia where hemiplegia or other paralytic lesions develop, cortical haemorrhages in the motor areas have probably oc-

curred, with or without recovery. The high blood pressure associated with the disease predisposes to the occurrence of these haemorrhages, but since venesection has been revived, a reduction in the incidence of this lesion has been observed.

Heart.—The ventricles are usually contracted, the auricles full of dark blood, which does not clot readily, contrary to the condition of hyperinosis which existed during life. The heart muscle is fatty with tiny haemorrhages, necrosis and thrombi; it tears easily, and these changes may be a cause of death. Fatty heart is found especially in those cases which have been given large doses of chloroform and chloral, a fact which warns to care in the administration of these drugs. There may be subpericardial haemorrhages.

Chemical Findings.

Blood:—

1. Eclampsia is seldom associated with nitrogen retention in the blood, and where the N.P.N. is elevated, it is probably a result of kidney injury caused by the eclamptic outbreak.
2. The same holds true for blood urea N.
3. An increase in the uric acid and a decrease in the CO_2 combining power are the outstanding findings in the blood chemistry.
4. The blood sugar is usually within normal limits, but hypo and hyperglycaemia have been reported.
5. The blood lactic acid is increased.
6. The pH of the blood is normally 7.47 during the latter months of pregnancy, is not altered in the pre-eclamptic state, but reaches 7.28 during convulsions. The severity of the condition often seems to depend on the degree of acidosis.
7. Amino acids are normal, there being apparently no failure in deaminization.
8. The creatine and creatinine metatabolism are not markedly upset.
9. The basic radicles are normal except for a slight increase in P.
10. The fat and lipoids are as in normal pregnancy.
11. The blood colloids show a shifting in the protein fraction in favour of globulin, fibrinogen and euglobulin. Fibrinogen, which in normal pregnancy is 76 mg. /100 cc., becomes in mild eclampsia about 87 and in severe eclampsia about 100.
12. The albumin/globulin ratio, which is 2.6 in the non-pregnant and 0.81 at term, is still further decreased in eclampsia to about 0.21.

Urine.—

1. The urine is deeply coloured and of normal or even high specific gravity.
2. The daily urinary output is usually much diminished.
3. Albumin is present in great abundance in the severer cases, and is of two kinds, serum albumin and lactalbumin.
4. Hyaline and hyalo-granular casts are present in large numbers and are of small size.
5. Epithelial casts are comparatively infrequent.
6. Microscopic blood is rarely present though red blood cells are occasionally found.
7. The excretion of chlorides is usually impaired in this type of toxaemia, and in the course of the large diuresis that accompanies improvement, the amount of chloride passed in the urine greatly exceeds the amount ingested, showing that in the course of the disease a large store of chlorides has accumulated in the body.
8. Investigation of the capacity of the kidney for water excretion shows, in the great majority of cases, definite impairment.
9. The capacity for excreting nitrogenous waste products, in a fair pro-

portion of cases, shows no functional impairment. Though this capacity of the kidney need not necessarily be impaired, in the majority of cases some diminution in the capacity for excreting nitrogenous waste products is found. The degree of impairment is not usually very striking, but is most marked in those patients in whom albuminuria has been present for a considerable time. The urea N is lowered and the ammonia N raised.

10. The ratio of unoxidized/oxidized sulphur is greater than normal.
11. Ketone bodies are frequently present.
12. The diastatic index is often greatly increased, reaching 200-300. (Normal, 6.6 - 30).

The Management of the Pre-eclamptic State.

(a) **Prophylactic.**—One can speak of rational prophylaxis only where the cause of the disease is known. Hence in pre-eclampsia, our treatment is all empiric. Every pregnant woman should be considered a potential eclamptic, and a significant family or personal history should put the physician doubly on his guard. The main factors in prophylaxis are:—

(i) Increased rest.
(ii) Treatment of focal infection.
(iii) Care of elimination.
(iv) Very close observation.

A minimum scheme for observation is a urinalysis once a month during the first five months, twice a month during the next three months, once a week during the last month. The patient should be seen personally by her physician not less than once a month for the recording of blood pressure, and should be impressed with necessity of notifying him of any untoward symptoms which may occur in the interval.

(b) **Expectant.**—This depends to a great extent on the rapidity of onset of the symptoms and the actual state of the patient when first seen.

There are a certain number of cases where a patient may be examined and found normal, the urine containing no albumin, and in which within a week the urine may be loaded with albumin and a marked general oedema present. On further examination the blood pressure in these patients will be found to have risen from the normal to 180 mm. Hg. or over. When one meets with this type of case, the pregnancy should be terminated at once without any consideration of the viability of the foetus. But in a large number of cases the onset is more gradual, and even if not seen till fairly marked symptoms are present, the patients will probably give a history of not feeling well for some weeks and will have noticed a slight general oedema. It is with this type of case that expectant treatment is justifiable for a time, with the hope of improving the chances of life for the child.

It should consist of:

1. Hospitalization, if possible.
2. Absolute rest in bed, preferably isolated, but this is not absolutely essential.
3. *Diet.* Since in the majority of cases there is no marked impairment of nitrogenous excretion, it is not necessary to restrict proteins in the diet. It should be light, and may be chosen from the following: milk, bread and butter, rice, eggs and fruit. Salt should be excluded, since chloride excretion is poor. The quantity of fluid given should be limited to two pints in the twenty-four hours, since the giving of large quantities of fluid in these cases does not produce a corresponding diuresis, and consequently only tends to accumulate and increase oedema.
4. *Purgation.* The bowels should be regulated, preferably with saline aperients,—

   Mag. Sulph.
   Sod. Sulph. aa 1 or 2 oz.

   as may be required, should be given daily, aiming at getting two or

three loose actions in the twenty-four hours.

5. Sleep is very essential, and if the patient does not sleep well, the difficulty should be overcome by the use of sedatives, such as:—
   Chloral gr. xx-xxx
   Pot. Bromide gr. xv-xx
   which should be given nightly.
6. Close observation, noting—
   (a) Volume output of urine.
   (b) Amount of albumin.
   (c) Presence or absence of casts.
   (d) Height of N.P.N. (usually there is no retention).
   (e) Signs of liver involvement.
   Vomiting.
   Urobilinuria.
   Diminished liver volume.
   Bile in the urine.
   Jaundice.
   (f) Blood Pressure.

The chief guide as to how long the treatment may continue is the blood pressure; to this may be added the total quantity of urine passed in the twenty-four hours.

(c) **Radical.**—Apart from such treatment as has been suggested above, our chief means of averting the actual onset of fits is induction of labour. Even in cases in which the child is not viable, there should be no hesitation in inducing labour if the symptoms appear to warrant this course. The actual onset of eclampsia is attended by very considerable danger to the mother and the foetus, while on the other hand, when once the pregnancy has been safely terminated, a complete recovery is the rule, and further pregnancies are almost invariably possible.

1. The chief guide in recommending induction should be the blood pressure. If, when the case is first seen, the blood pressure is in the neighborhood of 180 mm. Hg., and if, after two or three days of treatment, no fall has occurred, induction should be performed irrespective of the stage of pregnancy. A moderate blood pressure of about 160 mm. Hg., which shows a steady rise from day to day in spite of treatment, is also an urgent indication for induction. If with rest and treatment the blood pressure shows a tendency to fall, it is justifiable to prolong the pregnancy with a view to improving the chances of the child, though such cases should be carefully watched.
2. If there are signs of liver involvement. These are vomiting, urobilinuria, diminished liver volume, bile in the urine, jaundice.
3. If, in rare cases, the N.P.N. rises markedly.
4. If there are eye symptoms, especially eye-ground changes.
5. If there are symptoms of increased intracranial pressure, as headache and vomiting, especially if they appear suddenly. Frequently these are the immediate precursors of convulsions
6. If it is believed that convulsions are inevitable.

The chemical examination in this condition, though valuable in determining the diagnosis, is of little prognostic importance. It is not as yet possible to foretell the onset of convulsions by any of the chemical methods at our disposal. In those cases of pre-eclampsia in which nitrogenous excretion is impaired, this function of the kidney should be examined from time to time, and any progressive deterioration should be regarded as an indication for inducing labour; a blood urea about 50 mg., a level which is infrequent in cases examined before the actual onset of fits, is also an indication for terminating the pregnancy.

There can be no doubt that in the present state of our knowledge, frequent determinations of the blood pressure are, when once the diagnosis has been established, of more value than chemical examinations. At the same time it has to be admitted that a few cases of eclampsia are on record in which no increase in the blood pressure

was found, and that there is no critical level of blood pressure at which fits invariably occur.

Methods Of Termination.

The methods of termination in common use are:—

1. Hydrostatic bags or bougies.
2. Caesarian Section.

Caesarian section is the only method to be considered when the toxaemic process is rapid and virulent, or where there are obstetrical difficulties, as in elderly primiparae, contracted pelvis, transverse positions, etc.

The usual method, however, is by the use of hydrostatic bags or bougies, supplemented by ergot or quinine per os.

It is characteristic of toxaemias to go into labour prematurely. If labour has already begun and no obstetrical difficulties are anticipated, it may be allowed to proceed without interference, and the patient watched closely for any signs necessitating rapid delivery.

After Treatment.

After delivery, careful observation of the amount of urine passed daily and the quantity of albumin present should be kept, and before the patient is allowed up a more detailed examination of the renal function should be carried out.

This should be repeated at the end of six months, and then again at the end of a year, and if the kidney function is found to be normal, as is usually the case, then the patient should be told that the probability is that the condition will not recur in the next pregnancy; but there should be no possibility of further pregnancy occurring before that year has elapsed.

Summary.

1. Pre-eclampsia is essentially eclampsia before the outbreak of convulsions or coma.
2. No theory of etiology so far suggested can be considered conclusive.
3. Of the theories in vogue at present, that of the toxic origin of eclampsia is most popular.
4. It is impossible clinically or chemically to determine who will develop convulsions and those who will go to term without further symptoms.
5. A positive diagnosis can be made only on post-partum progress.
6. The most important signs of pre-eclampsia are hypertension, albuminuria and oedema.
7. There is no constant pathological picture.
8. The kidney lesion is probably the result rather than the cause of the disease.
9. The chemical findings are fairly constant but not pathognomonic.
10. Every pregnant woman should be considered a potential pre-eclamptic and be subject to constant supervision.
11. Method of treatment depends largely on the rapidity of onset of symptoms and consists chiefly of absolute rest in bed, increased purgation and salt-free diet.
12. The best indication of improvement is a gradual fall in blood pressure.

THE NERVE-SUPPLY OF CEREBRAL BLOOD-VESSELS AND ITS RELATION TO EPILEPSY

ERIC A. LINELL, M.D. (Manch.).

Anatomy.

Until recent years, intracranial blood-vessels were believed to be devoid of vaso-motor nerves and variations in calibre of these vessels was thought to occur passively as a result of fluctuations in the general arterial blood pressure.

With improvements in the technique of histological staining, nerves have been demonstrated accompanying the larger cerebral blood-vessels and the branches of these vessels in the pia mater, but it is only within the last few years that reliable observations of nerves following the terminal branches of the pial arteries into the brain tissue have been published. Hassin, writing in 1929,[1] stated that, although the arteries of the pia mater were richly supplied with nerve fibres, the vessels of the cerebral parenchyma were not innervated. He concluded, therefore, that the control of blood-flow through the intracerebral vessels was mediated by the vaso-motor nerves of the pial arteries.

Clark,[2] using Ranson's pyridine silver method of staining, was among the first to publish, in the same year, convincing histological evidence of nerve fibres accompanying the terminal branches of the pial arteries into the nervous tissue of the medulla and spinal cord. He believed that these intracerebral vascular nerves were of two types: (1) thin, non-medullated fibres continuous with the plexus round the pial arteries, and (2) thicker non-medullated fibres. The former, he suggested, were the terminations of sympathetic fibres, and he believed that they ended in the muscle coat of the vessels. He thought that the latter type were probably branches of cranial nerves and had their endings in the adventitial vascular coat.

In 1932 Penfield,[3] using Bielschowsky's silver stain, confirmed Clark's finding of intracerebral vascular nerves. Although he did not commit himself as to the different origin of the thick and the thin non-medullated fibres found, he stated that a proportion of the fibres did not degenerate after removal of the cervical sympathetic trunk, and therefore, presumably, were not sympathetic in origin.

From these anatomical observations it may be concluded that both the nerves of the pial arteries and their terminal branches in the brain tissue arise from two sources—from nerve-cells in the cervical sympathetic ganglia and from cranial nerves.

Physiology.

Cushing observed in 1902 that the application of adrenalin to the cortex of the brain caused a local constriction of the pial vessels, and that the same local constriction was produced by electrical stimulation of the cortex.

In 1928, Forbes and Wolff[4] published their important observations on the vaso-motor control of the vessels of the pia mater. Working with cats, they removed a portion of the skull over a cerebral hemisphere, and replaced the bony defect by a transparent window, so that they could observe changes in calibre of the pial arteries over prolonged periods. They found that electrical stimulation of the cervical sympathetic trunk caused constriction of the arteries of the pia mater over the corresponding hemisphere, and that this was not accompanied by any lowering of the animal's systemic blood-pressure. They therefore concluded that this change in calibre was the re-

sult of active vaso-constriction of the arteries observed. Electrical stimulation of the central cut end of the vagus nerve caused, conversely, an active dilatation of the pial arteries, which was unaccompanied by any rise in the systemic blood-pressure.

In the following year, Talbot, Wolff and Cobb[5] showed that following unilateral section of the cervical sympathetic trunk, in the cat, there was a marked dilatation of vessels throughout the corresponding cerebral hemisphere. They showed this by injecting the vessels with carmine and then "clearing" the hemispheres. The dilated capillary bed is seen through the transparent brain tissue in the hemisphere of which the vessels had been deprived of their vaso-constrictor fibres by previous section of the cervical sympathetic trunk.

It would seem, therefore, from these experiments, that the cervical sympathetic trunk carries vaso-constrictor nerve fibres to the pial, and probably to the intracerebral arteries, and that the vagus nerve carries fibres which, on stimulation, cause active dilatation of the same vessels. The anatomical pathway of vagal dilator impulses to cerebral vessels has been suggested in recent articles by Cobb and Finesinger[6] and Chorobski and Penfield.[7]

The Relationship of Cortical Vascular Changes to Epilepsy.

Scattered references through the literature have continually suggested the spasmodic contraction of cortical arteries as an accompaniment of an epileptic seizure. Hughlings Jackson in 1863 observed spasm of the arteries of the retina during an epileptic fit.

Kennedy, in 1923, and Foerster, in 1926, were able to report their observations of the cerebral cortex of patients who suffered from a seizure while on the operating table, with the cerebral cortex exposed. Both observers reported pallor of the cortex during the first stage of the fit, corresponding roughly in duration with the tonic stage. During the succeeding clonic stage of the seizure, the vessels of the cortex become markedly congested. It seems probable, therefore, that during an epileptic attack active constriction of pial, and probably of intracerebral, vesseds is followed by active dilatation of these vessels.

That local vaso-dilatation of the cerebral cortex may occur in response to functional activity is suggested by a case reported by Fulton.[8] His patient had an arterio-venous aneurysm in the neighbourhood of the visual area of his cerebral cortex. The thrill of the aneurysm could be heard through a stethoscope applied to the skull over this area. The thrill was definitely increased in volume when the patient was asked to increase the functional activity of his visual area by reading.

Local vaso-constriction of cortical vessels in relation to epileptic seizures has been strongly suggested by Penfield[9] in a recent publication. He has observed the exposed cerebral cortex of epileptic patients over prolonged periods, and has frequently observed an attack to be initiated by a definite spasmodic constriction of the pial artery with an accompanying local pallor of the cerebral cortex. If this constriction does not occur spontaneously, it can frequently be produced by electrical stimulation of the pia mater, the vaso-constriction being followed by pallor and the production of a fit. Sometimes this author has observed vaso-constriction to occur after the seizure has passed off. He concludes that "the one constant, visible phenomenon during an epileptic seizure is cessation of arterial pulsation. Pallor may be present during a seizure, but more often follows it. After the attack the cerebral arteries pulsate violently, and, I believe, more rapidly than before operation. The colour becomes bright red, and arteries which were not seen to pulsate before the seizure now begin to do so visibly."

In view of these observed cortical vascular changes in epilepsy, it would

be reasonable to expect to find some histological changes in the cortex of the epileptic brain referable to such a vascular upset. Spelmeyer[10] in 1930 reported small focal areas of destruction of cortical nerve cells in Ammon's horn in the brains of epileptics.

In a case of epilepsy recently coming to autopsy from the Toronto General Hospital, small focal bulging areas were found in the cerebral cortex after fixation of the brain. Histologically, the nerve cells were shrunken in these areas, and the general appearance was suggestive of small foci of oedema of the cerebral cortex. It seems likely that vaso-constriction of an area of cerebral cortex followed by vaso-dilatation would result in an oedema, which would probably be transient, in this area.

Summary:

In this review of recent investigations into the anatomy and physiology of cerebral vascular nerves, the relationship of vascular changes to epilepsy has been considered. The only justifiable conclusion which may be drawn is that vaso-motor changes accompany the seizure in the brain of an epileptic.

If epilepsy were caused by vaso-motor spasm, it should be curable by removal of the cervical sympathetic trunks, which is not the case, although occasional cases of marked improvement have followed this operative procedure (Penfield[11]).

Reports of the results of direct operative attack on the vessels which have been noted to show spasmodic phenomena, will be followed with great interest.

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(7) Chorobski and Penfield—Arch. Neurol. Psych. Page 1257. Vol. 29, 1932.
(8) Fulton—Brain. Page 310. Vol. 51, 1928.
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WHAT EVERY WOMAN DOESN'T KNOW—HOW TO GIVE COD LIVER OIL.

What Every Woman Doesn't Know is that psychology is more important than flavouring in persuading children to take cod liver oil. Some mothers fail to realize, so great is their own distaste for cod liver oil, that most babies will not only take the oil if properly given, but will actually enjoy it. Proof of this is seen in orphanages and pediatric hospitals where cod liver oil is administered as a food in a matter of fact manner, with the results that refusals are rarely encountered.

The mother who wrinkles her nose and "makes a face" of disgust as she measures out cod liver oil is almost certain to set the pattern for similar behaviour on the part of her baby.

Most babies can be taught to take the pure oil if, as Eliot points out, the mother looks on it with favour and no unpleasant associations are attached to it. If the mother herself takes some of the oil, the child is further encouraged.

The dose of cod liver oil may be followed by orange juice, but if administered at an early age, usually no vehicle is required. The oil should not be mixed with the milk or the cereal feeding unless allowance is made for the oil which clings to the bottle or the bowl.

Mead's 10 D Cod Liver Oil is made from Mead's Newfoundland Cod Liver Oil. In cases of fat intolerance, the former has an advantage, since it can be given in 1-3rd to half the usual cod liver oil dosage.

(To be continued).

LYMPHATIC LEUKEMIA

By J. B. AIKEN, M.D.
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Leukemia may be defined as a pathological condition in which there is extensive hyperplasia of the leukoblastic tissues with appearance in the blood of abnormal white cells. Two forms, lymphoid and myeloid, are distinguished. In Lymphatic Leukemia the cells show qualitative and quantitative changes. However, some cases with leukemic blood show no evidence of leukemia on the autopsy table; this perhaps represents an abnormal response of the bone marrow. Also some cases with a normal blood picture, or only a relative lymphocytosis, with little qualitative change in the blood, show at autopsy the typical findings of a lymphatic leukemia. Also in acute cases of the disease one can, only with difficulty, or perhaps not at all, distinguish clinically between the lymphoid and myeloid types. Thus, we cannot say that leukemia is a definite entity, but that it is a disease characterized by several states. These states, in which the manifestations are chiefly lymphoid, will be discussed in this paper.

History.

In 1845 Bennet and Virchow independently described cases of splenic enlargement with increased white blood count and whitish appearance of the blood. Bennet called it leucocythemia, while Virchow referred to it as leukemia. They did not, however, make the observation that these cells differed qualitatively from those found in sepsis. Virchow later distinguished a lymphatic and a splenic form in which the lymph glands or spleen were enlarged respectively. In 1890 Neumann proposed the term myelogenous for a third type. In 1891 Ehrlich, by means of differential staining, distinguished two types of cells: granulocytes and non-granular cells of lymphatic origin. The granular cells were shown to be derived from the bone marrow. This resulted in a division into myeloid and lymphoid types, which remains to-day.

Incidence.

Lymphatic Leukemia is widely distributed in the animal kingdom. It frequently occurs in fowl, and has been described in cattle, dogs, rats and mice. In human beings it is relatively infrequent, one case occurring in every 1,000 hospital admissions. Chronic lymphatic leukemia occurs between the ages of 45 and 60; acute lymphatic leukemia occurring in individuals less than 25 years old. A familial character has been ascribed to leukemia. The incidence of the disease in males is twice that in females.

Classification.

Blood cannot be regarded as a tissue with fluid matrix, but as a secretion dependent upon the haematopoietic tissues (Piney). Variations in the blood picture are accompanied, or rather preceded, by overgrowth of the haematopoietic tissues with the appearance of immature cells of their own type in the blood. Piney refers to these as leukoses. He makes a classification into leukosis of myeloid and lymphoid types. But since these cells may not appear in the blood, a further sub-division into leukemic and aleukemic forms is necessitated. His classification avoids the use of the term leukemia where there is no increase in white blood cells, but a post mortem picture of a leukosis. There is further division into acute and chronic types. MacCallum puts forth a classification, a modification of Sternberg's classification. A combination of the two classifications,

with the reticular cell as the primitive origin of the cells, would attempt to explain the development of all these conditions upon a monophyletic basis.

Clinical History and Findings.

Chronic lymphatic leukemia (Leukosis)—The patient, an adult, complains of swelling in the neck, axilla and groin, or of symptoms due to tonsillar or mediastinal involvement. Weakness and fatigue are also frequent complaints. Physical examination shows generalized enlargement of lymph glands and a palpable spleen. The lymph glands are freely movable and hard, with no evidence of fluctuation. The skin may occasionally show papular eruptions. The diagnosis is made on the blood examination. The findings usually correspond with the magnitude of the organic lesions. A great increase in white blood cells of the lymphocyte variety is found, the count averaging between 100,000 and 200,000 per cubic mm. The small lymphocyte predominates, but large lymphocytes can also be found. These cells are fragile, as shown by the presence of smudges (irregular masses of chromatin material). A secondary type of anaemia is also present, with a few normoblasts. Some primitive myeloid cells may also be found, but these are few. Increase in eosinophiles may be present. The chronicity of the case is shown by the predominance of the small lymphocytes. An increase in the number of large lymphocytes and the presence of many lymphoblasts indicates a rapidly approaching fatal end. The general condition of the patient may be fair, with only slight anaemia, but towards the end anaemia is quite marked with normoblasts in the blood. Weakness and cachexia then progress rapidly. The disease is progressive and invariably leads to death within two to ten years. There is a tendency for spontaneous remissions, during which the patient temporarily improves. Radiation therapy may prolong the patient's life for a short time.

Chronic Aleukemic Lymphatic Leukemia (Aleukemic Lymphadenosis) — This form of the disease is much less common than the leukemic type, but the clinical signs are similar. The white blood count is about normal. Skin involvement is more common. Diagnosis by study of the blood may be difficult, but a relative lymphocytosis of 70% or over, with a normal, or even lowered, white blood count, is very suggestive.

Acute Leukemia (Leukosis)—These, both lymphoid and myeloid, occur usually in children and adolescents, chronic leukemias being very rare in childhood. The patient becomes ill with a high fever, tonsillar ulceration, petechial haemorrhages, and a tendency to bleed from mucous surfaces. There is usually a history of an acute upper respiratory infection. A rapidly progressing anaemia is found, which, along with the fever, leads to a suspecion of a septicaemic condition. Splenomegaly and lymph gland enlargement are absent. The disease progresses rapidly, and death occurs within a few days to a month. The blood examination shows an increase in white cells, which are seen in the smear to be lymphoblasts. The count may be 10,000 on the first occasion, and this leukopenia may persist or rise to a count between 20,000 and 100,000 per cubic mm. The count is usually lower than in the chronic type. These cells are difficult to distinguish from myeloblasts in acute myeloid leukosis, but this differentiation is unimportant clinically. The oxydase reaction usually aids in distinction, but very immature myeloblasts do not give this reaction. Acute septic infections may give similar blood pictures, and sometimes the clinical course is the only means of differentiation. In these cases of lymphocytosis with septic sore throat recovery occurs and the blood picture returns to normal.

Pathology.

The pathological picture is that of a diffuse involvement of the blood form-

ing organs, with secondary invasion of other tissues. Grandular enlargement rather than splenomegaly is characteristic. In the chronic form the lymphoid tissue is not involved equally all over the body. In the acute type the process is the same as in the chronic, but not as well marked. This is probably due to the shorter duration of the condition.

The lymph glands in the chronic form are enlarged and composed of soft, grayish white cellular tissue. The disease appears in one or more groups, spreading to other groups, but involving all the glands of a group simultaneously. The enlarged nodes may be discrete or matted together by inflammatory reaction. Their structure is largely obliterated. Microscopic section shows the normal architecture practically completely replaced by the growth of small lymphocytic cells and a relatively small number of large lymphocytic cells. In the acute disease the same grayish white tissue is seen, but it is rather softer, shows a tendency to small haemorrhages, and in microscopic section the usual structure of the lymph gland can often be made out. Submucous lymphoid tissue shows the same alteration as the lymph nodes

The spleen is moderately enlarged, and the Malpighian bodies are much enlarged. In more chronic cases the organ is a grayish mass of tissue in which the Malpighian bodies cannot be discerned. Microscopic examination shows the pulp spaces, venules and Malpighian bodies overrun with lymphocytes. This infiltration may be so dense that the trabeculae form the only clue as to the identity of the tissue. Small areas of infarction and necrosis are seen, while minute areas of fibrosis occur in chronic cases.

In the bone marrow the process begins in many small hyperplastic foci, which enlarge and coalesce, and finally spread to all parts of the skeleton. The red marrow occupies a greater portion of the marrow cavity than usual, to the detriment of the fatty marrow. It is firm, has a reddish gray appearance, and may show small areas of haemorrhage and necrosis. Histologically lymphatic tissue replaces the fat tissue and infiltrates the myeloid tissue, but in addition the myeloid tissue itself is greatly increased in amount. There may be some absorption of bony trabeculae, but there is no bone destruction.

Outside of the haematopoietic organs, the liver is the chief tissue involved. It shows fatty degeneration, but the most important change is infiltration with lymphocytes. This consists of dense miliary accumulations in the periportal areas, with some cellular infiltration in the rest of the liver tissue. Any organ may show lymphatic infiltration. The kidney often shows focal and diffuse infiltrations. Skin nodules may appear as papular eruptions, especially on face and neck, and the skin may show a marked reaction in the form of other skin lesions.

Effect Of Radiation Therapy.

Radiation, which is the chief method of treatment, produces marked changes in the clinical and pathological pictures. X-ray or radium cause a decrease in size of the lymph glands and spleen and improvement in the general condition of the patient. Lymphatic tissue is much more injured by X-rays than granulocytic or erythropoietic tissue, and, in fact, practically all the lymphocytes can be removed from the blood with little effect on the other cells. The effect does not set in at once, and only reaches its greatest intensity hours or days later. Repeated exposure to X-rays causes anaemia and leukopenia. All elements of the bone marrow are depressed even to the stage of aplastic anemia. The lymph glands histologically show a similar picture to that described before. The striking changes are seen in the bone marrow with repeated doses of X-ray. The lymphatic tissue and even the myeloid and erythropoietic tissues are decreased and replaced by a fine fibrous connective tissue.

Reticular Cell.

A. Lymphoid Hyperplasia.
   1. With Leukemic blood.
      (Acute Lymphoid Leukemia).
      (Chronic Lymphoid Leukemia).
   2. Leukemic blood with tumours.
      (Leukosarcoma).
   3. Without Leukemic blood.
      (Tumours of Bone Marrow—
      Lymphoid and Plasma Cell
      Myeloma).
      (Aleukemic Leukemia).
      (Lymphosarcoma).
      (Status Lymphaticus).

B. Myeloid Hyperplasia.
   1. With Leukemic blood.
      (Acute Myeloid Leukemia).
      (Chronic Myeloid Leukemia).
   2. Leukemic blood with Tumours of
      Myeloid Tissue.
      (Chlormyelosarcoma).
   3. Without Leukemic blood.
      (Tumours of Bone Marrow).
      (Myeloid Myeloma).

C. Reticulo-endothelial Hyperplasia.
   1. Leukemic.
      (Monocytic Leukemia).
   2. Aleukemic.
      (Hodgkin's Disease).

D. Erythroid Hyperplasia.
   1. Erythremia.

MEDICAL SOCIETY NOTES.

Medical At-Home.

Royal York Hotel, Jan. 26th, 1934.

The Med. At-Home this year is being anticipated as one of the big events of Toronto's social season. The committee has secured the Wright Brothers and their orchestra, and contrary to other reports, this orchestra is still intact; and their music, together with many other features, point toward a great night for Meds.

Owing to the large attendance last year and the growing enthusiasm this year, it would be well to get your tickets and reservations early.

Open Meeting of the Medical Society.

The first Open Meeting this year will be held on Wednesday, December 13th. The speaker will be Dr. J. G. Fitzgerald.

Daffydil.

This year Daffydil will be under the direction of the following committee:

Chairman.....H. V. Cranfield, '34
Vice-Chairman and Secretary.....
.....H. M. Coleman, '34
Musical Director.....Bruce Tovee, '38
Treasurer ..... John C. Steer, '34
Property Manager..K. C. Charron, '34
Assistant Property Manager.....
.....R. H. Hamilton, '36
Publicity Manager .... F. C. Heal, '35
Banquet ..... R. S. Chenoweth, '35
Ladies' Representative .....
.....Miss J. A. McGeachy, '37
Sixth Year Representative.....
.....Ian W. Davidson
Fifth Year Representative.....
.....J. D. Hamilton
Fourth Year Representative.....
.....W. H. K. Ross
Third Year Representative.....
.....W. T. Mustard
Second Year Representative.....
.....J. H. Baillie
First Year Representative.....
.....M. H. Little

Editor of Epistaxis..E. J. Delorme, '35
Assistant Editors.....F. P. Dewar, '36
E. V. Anten, '37
Business Manager .. R. V. Chapple,'34

Daffydil will be held in Hart House Theatre as usual, and the dates have been announced as February 15th and 16th. This is one of the major functions of the year to which we all look forward with the greatest anticipation, and we may feel confident that the above committee will provide entertainment for us rivalling the best efforts of the past.

BOOK REVIEWS

HIGHWAYS TO HEALTH: Donald Y. Solandt, M.A., M.D., '33. Ryerson Press.

The appearance of this book when Dr. Solant had barely finished his highly successful graduating year was a complete surprise to all his friends. But to his class-mates who found the worries of sixth year Medicine occupation enough, it seemed almost incredible that Don. could have completed so ambitious an enterprise without a ruffle in his habitual composure.

The book is designed for use as a text in public schools, or in the lower years of high schools. As Canadian books of the type are notably scarce, a demand for it may be reasonably anticipated in Provinces other than Ontario where an authorized text is in use. Dr. Solandt has followed, to a certain extent, the general plan of the Ontario Health Book, which is indeed a worthy model. He and the publishers, however, have achieved improvement in at least two respects, namely, with regard to illustrations and printing. The illustrations are very plentiful, and well designed to stimulate interest. Many of them are pleasing examples of artistic photography, and (we suspect) from Dr. Solandt's own camera.

The language is simple throughout, and the analogies are well chosen, and show considerable experience in popular scientific writing. The faults of the book are of a minor character and scarcely worth mentioning. Perhaps the worst fault is the somewhat platitudinous nature of introductory paragraphs. Then again, though we realize that an educational book must be conservative, there is no doubt that in referring to the many affairs of personal life, such as exercise, habits and study, where science does not yet make very definite pronouncement, the author might have made the reading more interesting if he had not clung so closely to time-honoured and orthodox views. All mention of sex hygiene is cannily avoided, and quite properly, perhaps.

We heartily congratulate Dr. Solandt on a very creditable achievement.

J.K.W.F.

THE STUDY OF ANATOMY: Written for Medical Students by S. E. Whitnall. Second Edition. Rewritten and Enlarged. London: Edward Arnold & Co.; Toronto, McMillan Co. of Canada; Baltimore, Wm. Wood & Co.

This little volume of ninety-three pages is most interesting and also entertaining. It is written in Professor Whitnall's characteristic style, with a fluent and facile use of words which makes its reading very easy. It is neither prosy nor dull, for it possesses attractive metaphors, apt and unexpected similes, and an imaginative type of expression which holds and stimulates the reader.

There is much of very sound practical advice which is of use not only to medical students, but is invaluable also to many others. As its title indicates, the volume is intended to show the proper way in which anatomy should be studied and learned, and many valuable suggestions are made which can be applied to teacher as well as student in this subject.

Throughout the book the point is frequently emphasized that the ultimate aim of the study of anatomy is not just to acquire a special technical knowledge of the cadaver, but is the application of this knowledge to the thorough understanding of all the manifold structures in the living body.

The various aspects of the Science of Anatomy are explained, with illustrative examples cited. The principles involved in this science and its relations to other subjects are briefly and

entertainingly stated, so that the reader may acquire the proper mental perspective.

In the latter part of the book are found short sections reviewing such topics as books for collateral reading on the subject, the relations of teacher and student, and some very common sense rules on the writing of examinations. Concluding the work is a bibliography which should be of much value in stimulating further reading on the subject. The book is distinctly inspirational in its effect, and should be recommended to all medical students early in their course.

James C. Watt, M.A., M.D.,
Professor of Anatomy,
University of Toronto.

THE ARCHES OF THE YEARS: By Halliday Sutherland. Geoffrey Bles. $3.00.

The Arches of the Years is the story of the life of a Scottish doctor. Both the life and the manner of recounting it are unusually interesting. It is not an orderly autobiography; only the highlights of life are sketched, and those in a delightfully disjointed fashion. One can pick it up and begin reading anywhere and find it instantly absorbing.

The author's career began in a not unfamiliar manner. He failed in Anatomy. His father, who was himself a doctor, at once banished the boy to Spain for three months to visit his uncle and to study Anatomy. On his return he passed his examination successfully, and went on to the study of Pathology under George Hamilton, who left a great impression on him. During his vacations he had many interesting adventures, including a taste of whaling north of the Shetland Islands.

After graduation he returned to Spain as assistant to his uncle. His descriptions of the life and customs of Spain, as he saw them, are fascinating. He found those two great Spanish institutions, the Senorita and the bull fight, intensely interesting. His account of the spectacle of a great bull fight and of the life of a torero are exceptionally vivid. He even pursued his interest in bull fighting to the extent of having a try at it himself.

Returning from Spain, he undertook a variety of jobs. First he spent a short time on the staff of a mental hospital, next he had charge of a sanitarium, and, finally, while working in one of the London hospitals, he founded an open-air school for tuberculous children. It was the first of its kind in England.

During the war Dr. Sutherland served as a naval surgeon, for a time in the South Seas, and later on the south coast of England.

To the student who is wearied by the constant round of lectures, labs. and clinics, this book brings a new vision of the adventures that may be encountered in the practice of his chosen profession.

O. M. S.

RADIO ACTIVITY: ITS HISTORICAL DEVELOPMENT

By VERA J. WINSTON, '34.

With the twentieth century came an entirely new field of knowledge which, although beginning as a pure science, has proven of value in its application to medicine. Little did the world of science realize the portent of Roentgen's discovery in 1895 of a new type of radiation which he called the X-Ray.

He found that the production of X-Rays in a vacuum tube was accompanied by a strong phosphorescence of the glass, and it occurred to several investigators that ordinary substances made phosphorescent by visible light might emit a penetrating radiation similar to X-Rays. Following this idea H. Becquerel, a distinguished French physicist, placed, among other substances, a phosphorescent compound of uranium in black paper beneath a photographic plate. A weak photographic effect was obtained. This was shown to be due to a penetrating radiation capable of passing through sheets of matter opaque to ordinary light. At first he thought that the uranium rays were produced as a result of exposing the salt to light, but experiment showed that salts kept for several months in the dark continued to give off these rays. Further investigation showed that this photographic action was exhibited by all compounds of uranium being peculiar to the metal itself and having nothing to do with phosphorescence.

Madame Curie became interested in this new phenomenon and she resolved to undertake a special study of it. Being primarily a mathematician, she set out to measure the phenomenon with precision. To accomplish this, she used that property of the rays which enabled them to discharge an electroscope. Her determination showed that the emission of the rays is an atomic property of the uranium whatever were the chemical or physical conditions of the salt. It occurred to her that there might be other substances possessing this remarkable property. She soon found that substances containing Thorium behaved in a similar way and that this behaviour depended similarly on an atomic property of Thorium.

She proceeded to examine a number of minerals, and found that these containing Uranium and Thorium were radio active. But their activity was greater than could be accounted for by the quantities of these substances present. There must be, she thought, some unknown substance in these minerals. Thereupon she urged Pierre Curie, her husband, who at that time was professor of physics at the Sorbonne, to assist in the study of this new element.

Since they did not know any of the chemical properties of the latter, but only that the substance emits rays, it was by these rays that they had to search. They first undertook the analysis of a pitchblende from St. Joachimsthal. In a few months they could separate from it a substance accompanying the Bismuth which was much more active than Uranium. In 1898 they announced the existence of this new substance, which was called Polonium in honour of Madame Curie's native land. While engaged in this work on Polonium they also discovered that, accompanying the Barium separated from the Pitchblende, there was another new element to which was given the name of Radium.

Their task now was to separate these new substances as pure elements. The Austrian Government granted them the residues from the Pitchblende treatment plant in St. Joachimsthal.

While her husband continued to study the properties of the rays, Madame Curie devoted herself to the purification of Radium.

Finally, in 1910, this substance was isolated and found to show all the characters of a pure chemical body. She determined its atomic weight to be 226, and she also showed that it had a characteristic spectrum. Shortly afterwards a commission of scientific men of different countries was formed who agreed to take as a base an international standard consisting of a carefully weighed quantity of pure Radium salt. Madame Curie was appointed to prepare the primary standard, which is now deposited in the International Bureau of Weights and Measures at Sevres, near Paris.

Madame Curie obtained one decigramme Radium from one ton Pitchblende. The scarcity of this new substance has stimulated scientists to produce radiations, using very high voltages. At the California Institute of Pasadena, a large single X-Ray tube operating at 550,000 - 900,000 volts, has been used successfully, and a still larger tube of the same type is now being constructed for Biological experiments. The production of X-Rays as penetrating as Cosmic rays, with voltages of 16,000,000, is forecast by the researches of Drs. F. Lange and A. Brasch, of the University of Berlin. Working in a small valley between two mountain peaks at Mount Generoso, on the Swiss-Italian border, a place where thunderstorms occur frequently in summer and early autumn, these scientists have drawn electrical energy from the sky. These men succeeded in building a tube, known as the Lange Brasch X-Ray tube, which has been tested at 2,600,000 volts. These X-Rays are the most powerful yet produced. It is to be hoped that in the near future rays will be produced which will be equivalent to many times the Radium now available for medical and industrial purposes.

Thus a new science has been developed which has proved of great importance in physics, upsetting our idea of the permanency of matter. It showed for the first time that the atoms of certain of the elements are not permanently stable, but break up, the disintegration being accompanied by the expulsion of intense radiations. With this science also the foundation for a new method of chemical analysis has been laid whereby a substance is now examined for radio activity by a very delicate electroscope. And, finally, in its application to medicine, the discovery and use of Radium has played a great part in the alleviation of human suffering.

MEDICAL ATHLETICS.

Interfaculty Sports.

The Senior Meds. Rugby team have given their opponents a real battle this year. They were finally put out of the running by Senior School in a hair-raising last minute victory.

Junior Meds. came even closer to a title, being nosed out by Trinity by a half a point. Under the able coaching of Cy Young, an excellent team was developed, and only one game was lost all season.

In Box Lacrosse, the team has been giving everyone a tough tussle, but has only managed to pull out one tie. Keys has occupied the limelight with a record of 22 goals out of 27 scored.

The Volleyball team has been more successful, and has won the first two games against O.C.E. and S.P.S.

The Soccer team got off to a bad start this year by being forced to default the first game due to lack of players. However, they came back strongly, and gave a good account of themselves in later games.

In the Interfaculty track meet, Ma-

guire was first in the 440, and Magladery led the mile for a new record. Magladery, Maguire and Thompson were 1st, 2nd and 3rd respectively in the three mile race. In the harrier, Magladery was again first, Thompson 5th and McKerracher 6th.

Intercollegiate Sports.

The Medical representation on the Senior Rugby team has been much smaller than usual this year, but the lack of numbers has been more than made up by the distinction of the few who have played. Jack Sinclair, playing his last year in Intercollegiate Rugby, has maintained his position as the finest half-back in the league, and probably the best in Canada. His final performance against Queens left nothing to be desired. His kicking was wonderful, and the way he intercepted the Queen's passes was a joy to behold. Jack will be a great loss to the team and to the league, for he has always been a favourite, not only with his team mates, but with all the other .teams. His coolness in the heat of battle and his quick thinking at all times will be missed even more than his superlative kicking. With all his fame, Jack is the most modest of heroes, the most generous of team mates, and the best of sports. The whole faculty is proud of him, and wish him every possible success in the future.

It is a great pity that Sinc's final effort was marred by a serious injury to his running mate, Hal. Richardson. Hal. has been dogged by ill-fortune all season, injuries keeping him out of the game most of the time. We all wish him a most speedy recovery. Johnny Copp, the plunging half-back of the team, has also been prevented from showing his real form by a knee injury.

Meds. can also claim the greatest outside wing in the intercollegiate. No one who has seen Andy Henderson mowing down the opposing half backs would deny him that title. Holden of B. and M. also did good work for the team.

On Lou. Carroll's intermediates we have been ably represented by Doyle, Jackson, McKee, Newton and Williams. Jackson also distinguished himself by a very fine performance in his first game in senior company, against Queen's in the finals. On the Juniors, Caldwell, Gray, Machlachlan and Storey lent a hand in winning the title.

The championship English Rugger team this year looks almost like an all Meds. team. Among those who helped win the Intercollegiate championship are Caverhill, Elliot, Garner, Hilliard Kyle, Losier, Walsh, Whillans and Wilson.

The Faculty's enviable reputation on the track has been upheld this year by Magladery, Maguire, McKerracher and Thompson.

In Intercollegiate Soccer we were represented by Toone, and in Tennis by Tomlinson.

O. M. S.

Medettes.

At this early date, there is little to report in the way of actual prowess demonstrated and victories won in the realm of Medette Athletics, with the exception of Tennis.

This last sport, as usual, sneaked up on us unawares, and the Meds. held their customary eight o'clock tournament one morning to pick a team for interfaculty competition. Jean Davey, this and last year's intercollegiate star, Harriott Gates, Marg. Bickle, Marg. Daley, Jean Lang and Mary Albertson displayed their ability at the net by annexing six points for Medicine.

The managers of the Hockey, Basketball, Baseball and Swimming teams are all enthusiastic about their prospects in the coming season, and strongly urge all bashful athletes to turn out for practices, and the rest of the Meds. to support them by attending as many games as possible.

STAFF and GRADUATES

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BEHOLD A RUGGED INDIVIDUALIST

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